# Occupational transmission of hepatitis C virus resulting from use of the same supermarket meat slicer

**L. Bocket[1], S. Chevaliez[2], N. Talbodec[3], A. Sobaszek[4], J. M. Pawlotsky[2] and Y. Yazdanpanah[5,6,7]**

1) *Laboratoire de Virologie, Centre Hospitalier Universitaire de Lille,* 2) *Centre National de Référence des Hépatites B, C et Delta, Hôpital Henri Mondor Créteil,* 3) *Service d'Hépato gastro-entérologie, Centre Hospitalier de Tourcoing,* 4) *Institut de médecine du Travail, Centre Hospitalier Universitaire de Lille,* 5) *Service Universitaire des Maladies Infectieuses et du Voyageur, Centre Hospitalier de Tourcoing,* 6) *EA 2694, Faculté de Médecine de Lille and* 7) *Laboratoire de Recherches Economiques et Sociales, CNRS URA 362, Lille, France*

## Abstract

Tracing risk factors for acquiring hepatitis C virus (HCV) in an HCV-infected patient, the only identified risk was working at the same butcher's counter of a supermarket as another HCV-infected patient, using a common ham cutting machine, with frequent bleeding hand injuries. A phylogenetic analysis showed a high percentage of nucleotide homology between the two patients' strains.

**Keywords:** Hepatitis C virus, occupational transmission, phylogenetic analysis

**Original Submission:** 15 January 2010; **Revised Submission:** 18 March 2010; **Accepted:** 1 April 2010
Editor: D. Raoult
**Article published online:** 15 April 2010
*Clin Microbiol Infect* 2011; **17:** 238–241
10.1111/j.1469-0691.2010.03245.x

**Corresponding author:** Dr L. Bocket, Laboratoire de Virologie, Centre de Biologie-Pathologie, Av. du Pr J. Leclercq, Centre Hospitalier Universitaire de Lille, 59037 Lille Cedex, France
**E-mail: l-bocket@chru-lille.fr**

In developed countries, hepatitis C virus (HCV) is mainly transmitted through parenteral exposure, including drug use by injection and unsafe invasive therapeutic or nontherapeutic practices, blood transfusions before 1991 and occupational exposure among healthcare workers [1,2]. We report a case of likely HCV transmission between two women working at the same meat counter of a supermarket.

Ms Z and Ms C, both infected with chronic HCV, were followed in the hepato-gastroenterology unit of the Tourcoing Hospital, in France.

Ms Z (Patient 1), a 40 year-old woman, was diagnosed with HCV in September 2003. She reported a history of intravenous drug use in 1983. She was clinically asymptomatic and had normal transaminase levels at her first visit. A HCV-RNA test showed HCV genotype 1a with an estimated viral load of 5.67 $\log_{10}$ UI/mL. By June 2004, her transaminase levels had increased. Serum fibrosis markers showed a stage F1 liver fibrosis. She initiated treatment (peg-Interferon-Ribavirin), but stopped in October 2004 because of virological failure.

Ms C (Patient 2), a 44 year-old woman with no known risk factors for HCV, was diagnosed with HCV in September 2005. She was screened for HCV because of observed asthaenia with elevated transaminase levels. An HCV-RNA test showed HCV genotype 1a with an estimated viral load of 5.23 $\log_{10}$ UI/mL.

When asked for risk factors, Ms C revealed that she had worked at the same supermarket meat counter as Ms Z since 1988, using the same meat-slicing machine. The spiked trays of the meat slicers can quite easily cause haemorrhages in the hands of its users. Indeed, Ms C said that she and Ms Z frequently sustained hand injuries and did not always wear gloves. The two patients had no contact with each other outside of work.

HCV genotype was determined through direct sequencing of the 5′ noncoding region of the HCV genome, using the TRUGENE®HCV 5'NC Genotyping Kit (Siemens Bayer, Siemens Healthcare Diagnostics, Bayswater, Australia). We found the same HCV genotype for both patients (1a). Samples were sent to the French National Reference Center for Viral Hepatitis B, C, and Delta (Henri Mondor Hospital, Creteil). To analyze the genetic and phylogenetic relationship between the HCV strains isolated from the two patients, the three HCV genomic regions were PCR-amplified and sequenced: a portion of the nonstructural 5B (NS5B) coding region, 286 bp in length (nucleotide positions 8325–8610 according to strain HCV-H, accession number M67463); the region coding for the full-length E1 envelope glycoprotein, 551 bp in length; and the region coding for hypervariable region 1 (HVR1) of the E2 envelope glycoprotein, 81 bp in length.

Determination of the HCV genotype was performed by means of a phylogenetic analysis of NS5B sequences, which included prototype sequences from various subtypes of HCV genotypes 1–6 [3]. Genetic relatedness of HCV strains was

assessed by means of phylogenetic analyses of the three NS5B, E1 and HVR1 regions. Sequence alignments were performed by means of CLUSTAL W [4]. Phylogenetic relationships were deduced by means of DNADIST-NEIGHBOR, in PHYLIP, version 3.5 [5]. For Neighbour-joining analysis, a Kimura two-parameter distance matrix with a transition/transversion ratio (Ts/Tv) of 2.0 was used. Trees were drawn with NJ-PLOT software [6]. Their robustness was assessed by bootstrap analysis of 1000 replicates by means of the SEQBOOT in PHYLIP.

Phylogenetic analyses of these two samples confirmed their similarity: 95% of the nucleotides were identical in the NS5B and in the E1 regions, and 81% were identical in the HVR1 region. When we aligned the sequences from the E1 region of the two patients' HCV strains with the E1 region of 52 other sequences found in the French National Reference Center for Viral Hepatitis B, C and Delta laboratory, the strains appeared to be much closer to each other (95%) than to any of the others included: the mean identity with the other strains for the E1 region of the strain from patient 1 was 92.5% (range 88–95.1%) and, for the E1 region of the strain from patient 2, the mean identity was 92.1% (range 88–94%). The same results were observed for HVR1 region: 81% identity between the two patients' strains sequences in this region vs. 69% (range 50.6–75.3%) and 66.2% (range 51.8–75.3%) between the strains from patient 1 and patient 2 and 15 other laboratory HVR1 strain sequences, respectively (Figs 1 and 2).

These identity levels between the two patients' strains are very high in comparison with the previously published data. Sequence variability within genotype has been described in several studies. Rates of HCV sequence changes were found to be $1.44 \times 10^{-3}$ nucleotide changes per site per year over the whole genome, or 4.1 and $7.1 \times 10^{-4}$ changes per site

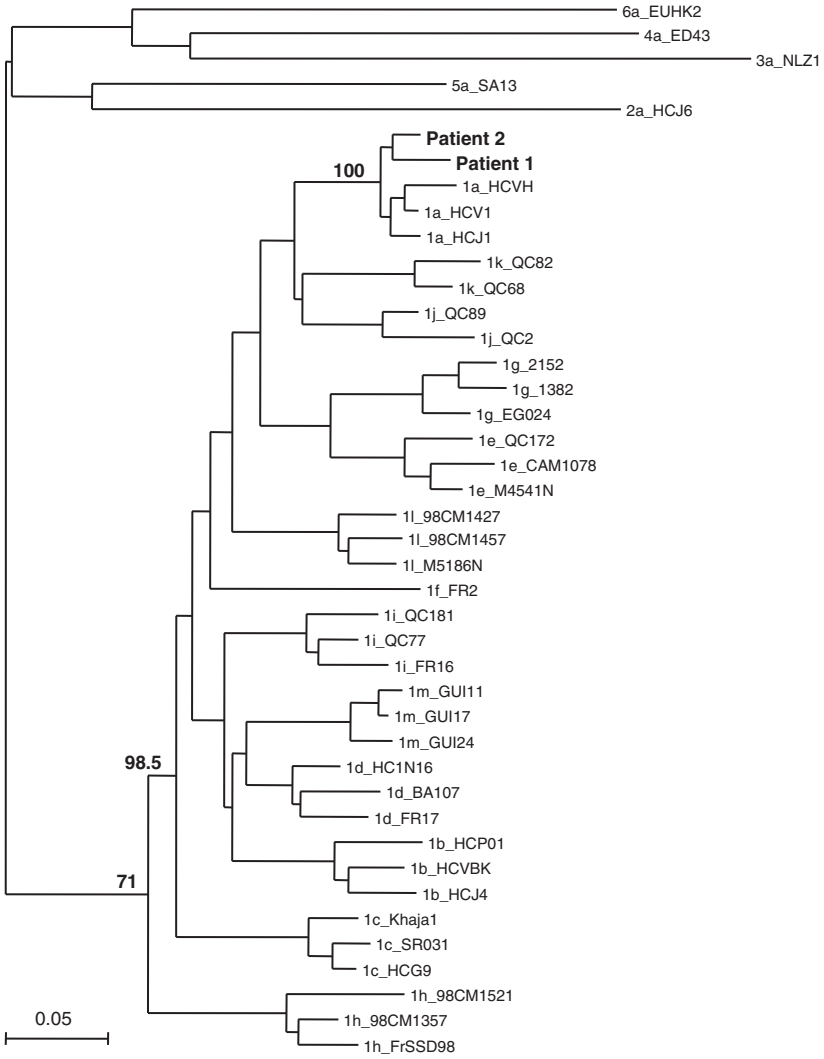


**FIG. 1.** Phylogenetic tree for the NS5B sequences (nucleotide positions 8325–8610) of the two patients in whom hepatitis C virus (HCV) transmission was suspected (patients 1 and 2, in bold), along with reference HCV strains of different genotypes and subtypes (the type and subtype are indicated before the strain identification letters and/or numbers). The nucleotide sequence of the NS5B gene of HCV strain EHK2 was used as an outgroup root.

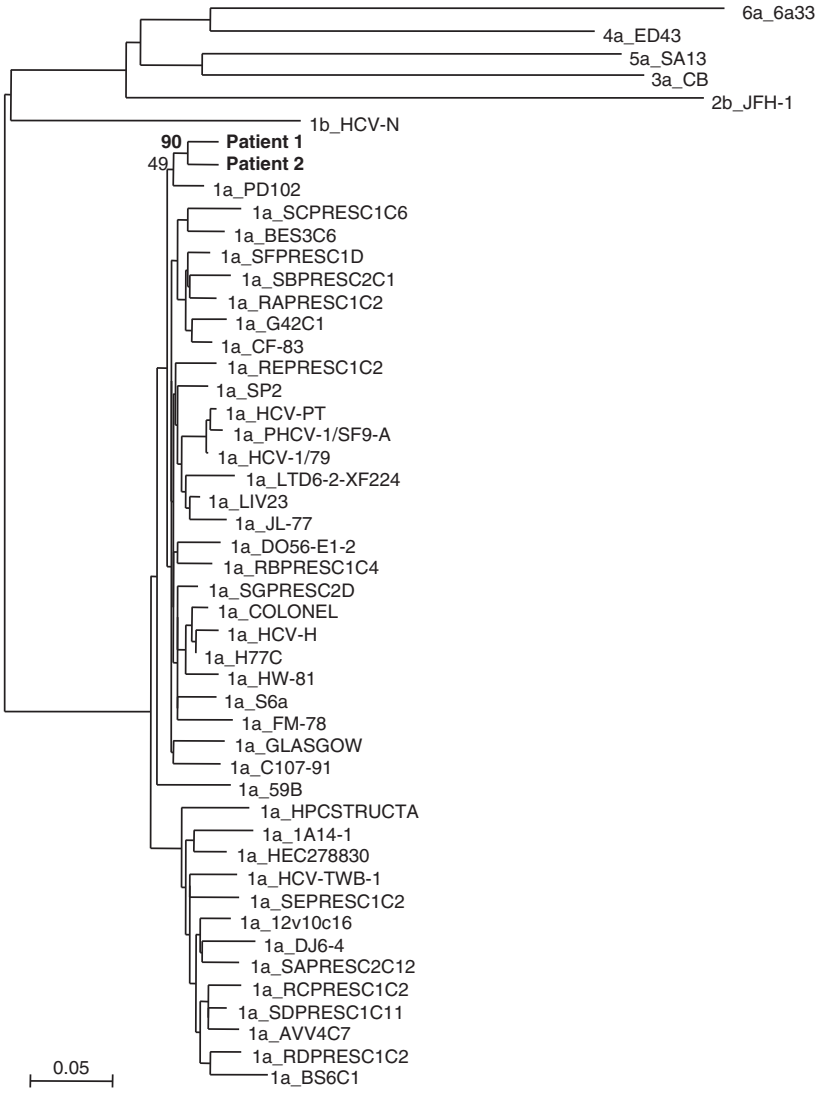


**FIG. 2.** Phylogenetic tree plotted with E1 envelope glycoprotein sequences from the two patients in whom an hepatitis C virus (HCV) transmission in a working context was suspected (patients 1 and 2, in bold) and with reference HCV strains of different genotypes and subtypes (the type and subtype are indicated before the strain identification letter and/or numbers).

per year in the NS5 and E1 region, respectively [7]. Moreover, in HVR1 of E2, the variability is much greater, as shown in an inter- and intra-individuals study of HVR1 evolutionary quasi-species analysis [8].

Phylogenetic analyses of both patients' HCV strains strongly suggest virus transmission from one to the other through occupational contamination. Ms C's medical history and as lack of other risk factors for HCV also act as strong arguments for this method of transmission.

This observation, reporting an unsuspected method of HCV transmission, is relevant in the actual context of HCV epidemiology. The prevalence of chronic carriers of HCV is not negligible. In France, it was recently estimated that 232 196 individuals (i.e. a prevalence of 0.53%) are chronically infected with HCV (i.e. HCV-RNA positive) [9]. No risk factors are identified in a high proportion of chronic careers of HCV. In Italy and France, the proportion of recently diagnosed patients with HCV infections without HCV infection risk factors has been estimated at 13–25% [10,11]. This unsuspected method of transmission could explain the lack of reported risk-factors for HCV infection among some infected patients.

Occupational transmission of HCV has been reported in the past but mainly among healthcare workers after needlestick injuries [2,12,13], with an average rate of HCV infection of 0.5% [14]. In a study on exposure to blood among paramedics, subjects were found to be at highest risk of exposure during contact to non-intact skin, which was more frequent than needlestick and mucocutaneous exposures [15]. Very little information is available on occupational transmission of HCV in non-healthcare settings. This observation emphasizes the fact that the promotion of

information on hepatitis C and other bloodborne viruses is also needed in non-healthcare settings, particularly in practices where sharp instruments such as slicers that are likely to cause hand injuries are used and shared by workers. In these practices, healthcare authorities should highlight the importance of wearing gloves and the prevention of cutting injuries.

## Acknowledgements

We are indebted to Dorothée Obach for her assistance in the preparation of the paper.

## Transparency Declaration

Y. Yazdanpanah received travel grants, honoraria for presentations at workshops and consultancy honoraria from Bristol-Myers Squibb, Boehringer Ingelheim, Gilead, Glaxo-SmithKline, Merck, Pfizer, Roche and Tibotec. All the other authors state the absence of dual/conflicting interests.

## References

1. Alberti A, Chemello L, Benvegnu L. Natural history of hepatitis C. *J Hepatol* 1999; 31 (suppl 1): 17–24.
2. Alter MJ. Epidemiology of hepatitis C virus infection. *World J Gastroenterol* 2007; 13: 2436–2441.
3. Simmonds P, Bukh J, Combet C *et al.* Consensus proposals for a unified system of nomenclature of hepatitis C virus genotypes. *Hepatology* 2005; 42: 962–973.
4. Thompson JD, Higgins DG, Gibson TJ. CLUSTAL W: improving the sensitivity of progressive multiple sequence alignment through sequence weighting, position-specific gap penalties and weight matrix choice. *Nucleic Acids Res* 1994; 22: 4673–4680.
5. Felsenstein J dbta. PHYLIP: phylogeny inference package, version 3.5c (computer program). Seattle, WA: Department of Genetics, University of Washington, 1993.
6. Perriere G, Gouy M. WWW-query: an on-line retrieval system for biological sequence banks. *Biochimie* 1996; 78: 364–369.
7. Simmonds P. Genetic diversity and evolution of hepatitis C virus–15 years on. *J Gen Virol* 2004; 85: 3173–3188.
8. Alfonso V, Flichman DM, Sookoian S, Mbayed VA, Campos RH. Evolutionary study of HVR1 of E2 in chronic hepatitis C virus infection. *J Gen Virol* 2004; 85: 39–46.
9. Anonymous. L'épidémiologie des hépatites B et C en France. *Bulletin Epidémiologique Hebdomadaire thématique* 2009; 20–21: 195.
10. Santantonio T, Medda E, Ferrari C *et al.* Risk factors and outcome among a large patient cohort with community-acquired acute hepatitis C in Italy. *Clin Infect Dis* 2006; 43: 1154–1159.
11. Brouard C, Delarocque-Astagneau E, Meffre C *et al.* Trends of hepatitis C screening in France through Rena-VHC and hepatology reference centres surveillance systems. *Bulletin Epidémiologique Hebdomadaire thématique* 2009; 20–21: 199.
12. Puro V, Shouval D. Hepatitis B, hepatitisC, and other blood-borne infections in healthcare workers-a VHPB symposium report- Rome, Italy, March 17–18, 2005. *J Viral Hepat* 2005; 14: 1–16.
13. Yazdanpanah Y, De Carli G, Migueres B *et al.* Risk factors for hepatitis C virus transmission to Health Care Workers after occupational exposure: a European case-control study. *Rev Epidemiol Sante Publique* 2006; 54 suppl 1: 1S23–1S31.
14. Jagger J, Puro V, De Carli G. Occupational transmission of hepatitis C virus. *JAMA* 2002; 288: 1469.
15. Leiss JK, Ratcliffe JM, Lyden JT *et al.* Blood exposure among paramedics: incidence rates from the national study to prevent blood exposure in paramedics. *Ann Epidemiol* 2006; 16: 720–725.

# Direct pathogen detection from swab samples using a new high-throughput sequencing technology

**H. Yongfeng*, Y. Fan*, D. Jie*, Y. Jian, Z. Ting, S. Lilian and Q. Jin**

*State Key Laboratory for Molecular Virology and Genetic Engineering, Institute of Pathogen Biology, Chinese Academy of Medical Sciences, Beijing, China*

## Abstract

The detection of emerging infectious diseases has been a continuing concern, especially with the novel influenza A (H1N1) viral pandemic of 2009. In the present study, we validated a 'second-generation' parallel sequencing platform for viral detection in swab samples collected during recent influenza virus infections in Beijing. This operation yielded millions of valid reads per sample and resulted in an almost complete spectrum of nucleotide information. Importantly, novel A (H1N1) and seasonal A (H3N2) influenza virus-derived sequences were detected without prior knowledge or use of genetic information in advance, suggesting that this approach could be a valuable tool for diagnosing emerging infectious diseases.

**Keywords:** Emerging infectious diseases, high-throughput sequencing technology, novel influenza A (H1N1) virus, Solexa system, viral detection

**Original Submission:** 1 March 2010; **Revised Submission:** 5 April 2010; **Accepted:** 6 April 2010
Editor: D. Raoult
**Article published online:** 15 April 2010
*Clin Microbiol Infect* 2011; **17:** 241–244
10.1111/j.1469-0691.2010.03246.x